



# ***STIC Search Report***

## ***Biotech-Chem Library***

**STIC Database Tracking Number: 134972**

**TO: Ralph J Gitomer  
Location: 3d65 / 3e71  
Art Unit: 1651  
Thursday, October 21, 2004**

**Case Serial Number: 09/980572**

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### **Search Notes**

=&gt; d his

(FILE 'HOME' ENTERED AT 14:01:47 ON 21 OCT 2004)

(FILE 'HCAPLUS' ENTERED AT 14:01:53 ON 21 OCT 2004)

L1 33 E4-6  
 E LONGLEY B/AU  
 L2 12 E3,E9  
 E LONGLEY J/AU  
 L3 418 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA  
 E SKIN DISEASE/CT  
 E E4+ALL  
 E E2  
 E E3+ALL  
 L4 70942 "SKIN, DISEASE"+OLD,NT/CT  
 L5 11 L4 AND L1-2  
 L6 24 L4 AND L3  
 E STEM CELL/CT  
 E E3+ALL  
 L7 30857 STEM CELL+NT/CT  
 E CELL/CT  
 E E3+ALL  
 L8 11056 CELL+OLD,NT/CT (L) STEM  
 L9 6 L5-6 AND L7-8

FILE 'STNGUIDE' ENTERED AT 14:06:51 ON 21 OCT 2004

(FILE 'WPIX' ENTERED AT 14:16:23 ON 21 OCT 2004)

L10 48555 (B12-A07 OR C12-A07 OR B14-N17? OR C14-N17?)/MC  
 E STEM CELL#/BI  
 E STEM CELL/BI  
 E STEM CELL/ABEX  
 L11 4846 (STEM (1A) CELL?)/BIX  
 L12 710 L10 AND L11  
 E LONGLEY B/AU  
 L13 3 E4  
 L14 1 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA  
 L15 1 L12 AND L13  
 L16 810 UYCO/PACO  
 L17 4 L12 AND (L14 OR L16)  
 L18 4 L15 OR L17

=&gt; b-hcap

(FILE 'HCAPLUS' ENTERED AT 14:22:30 ON 21 OCT 2004)

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FILE COVERS 1907 - 21 Oct 2004 VOL 141 ISS 17  
 FILE LAST UPDATED: 20 Oct 2004 (20041020/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L9 ANSWER 1 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN  
 AN 2002:674642 HCAPLUS  
 DN 137:210939  
 ED Entered STN: 06 Sep 2002  
 TI Methods of use of compounds which inhibit the stem cell factor signaling pathway  
 IN Longley, B. Jack  
 PA USA

Searched by Noble Jarrell

SO U.S. Pat. Appl. Publ., 22 pp., Cont.-in-part of U.S. Ser. No. 306,143.  
 CODEN: USXXCO  
 DT Patent  
 LA English  
 IC ICM C12Q001-00  
 NCL 435004000  
 CC 1-7 (Pharmacology)  
 Section cross-reference(s): 2

## FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2002123031	A1	20020905	US 1999-474478	19991229
	US 6576812	B1	20030610	US 1999-306143	19990506
	WO 2000067794	A1	20001116	WO 2000-US12405	20000505
	W:				
	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW:				
	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRAI	US 1999-306143	A2	19990506		
	US 1999-474478	A2	19991229		

## CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
US 2002123031	ICM	C12Q001-00
	NCL	435004000
US 2002123031	ECLA	C07K016/28A
US 6576812	ECLA	C07K016/28A

AB The invention provides a method of preventing or treating in a subject contact dermatitis which comprises administering to the subject an amount of a compound capable of inhibiting the stem cell factor signaling pathway effective to prevent or treat contact dermatitis so as to thereby prevent or treat contact dermatitis in the subject. The invention also provides a methods of preventing or treating in a subject hyperpigmentation, asthma, cutaneous inflammation, anaphylaxis and bronchospasm, mastocytosis, tumors which express activated kit, and conception.

ST stem cell factor signaling pathway inhibitor therapeutic; dermatitis hyperpigmentation asthma stem cell factor signaling pathway inhibitor; skin inflammation mastocytosis stem cell factor signaling pathway inhibitor; anaphylaxis bronchospasm tumor stem cell factor signaling pathway inhibitor; contraceptive stem cell factor signaling pathway inhibitor

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (IgA; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (IgD; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (IgE; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (IgG; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (IgM; stem cell factor signaling pathway inhibitors for)

IT Enzymes, biological studies  
 RL: BSU (Biological study, unclassified); BIOL (Biological study)  
 (SCF-cleaving; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (anal; stem cell factor signaling pathway inhibitors for)

IT Ligands  
 RL: BSU (Biological study, unclassified); BIOL (Biological study)  
 (binding; stem cell factor signaling pathway inhibitors for)

IT Dermatitis  
 (contact; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL  
 (Biological study); USES (Uses)  
 (fusion products; stem cell factor signaling pathway inhibitors for)

IT Digestive tract, neoplasm  
 (gastrointestinal stromal tumor, inhibitors; stem cell factor signaling  
 pathway inhibitors for)

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL  
 (Biological study); USES (Uses)  
 (humanized; stem cell factor signaling pathway inhibitors for)

IT Skin, disease  
 (hyperpigmentation; stem cell factor signaling pathway  
 inhibitors for)

IT Allergy  
 (hypersensitivity; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (injections, i.m.; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (injections, i.p.; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (injections, i.v.; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (injections, s.c.; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (intestinal; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (intralesional; stem cell factor signaling pathway inhibitors for)

IT Skin  
 (keratinocyte; stem cell factor signaling pathway inhibitors for)

IT Dimerization  
 (kit; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (liposomes; stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins  
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL  
 (Biological study); USES (Uses)  
 (monoclonal; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (mucosal; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (nasal; stem cell factor signaling pathway inhibitors for)

IT Gamete and Germ cell  
 (neoplasm, inhibitors; stem cell factor signaling pathway  
 inhibitors for)

IT Mast cell  
 (neoplasm, mastocytoma; stem cell factor signaling pathway inhibitors  
 for)

IT Drug delivery systems  
 (ophthalmic; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (oral; stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
 (otic; stem cell factor signaling pathway inhibitors for)

IT Bronchi, disease  
 (spasm; stem cell factor signaling pathway inhibitors for)

IT Allergy inhibitors  
 Anaphylaxis  
 Anti-inflammatory agents  
 Antiasthmatics  
 Antitumor agents  
 Asthma  
 Canis familiaris  
 Contraceptives  
 Dermatitis  
 Felis catus  
 Human  
 Mast cell  
 Melanocyte  
 Neoplasm  
 Peptidomimetics  
 Signal transduction, biological  
 Transformation, genetic  
 Urticaria  
 (stem cell factor signaling pathway inhibitors for)

IT Stem cell factor

Transgene  
c-Kit (protein)  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(stem cell factor signaling pathway inhibitors for)

IT Antibodies and Immunoglobulins  
Nucleic acids  
Organic compounds, biological studies  
Peptides, biological studies  
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL  
(Biological study); USES (Uses)  
(stem cell factor signaling pathway inhibitors for)

IT Drug delivery systems  
(topical; stem cell factor signaling pathway inhibitors for)

IT 9004-06-2, Elastase 97501-92-3, Chymase 138359-29-2, Kit tyrosine  
kinase  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(stem cell factor signaling pathway inhibitors for)

IT 454746-24-8 454746-25-9 454746-26-0 454746-27-1  
RL: PRP (Properties)  
(unclaimed nucleotide sequence; methods of use of compds. which inhibit  
the stem cell factor signaling pathway)

L9 ANSWER 2 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN  
AN 2002:539652 HCAPLUS  
DN 137:88453  
ED Entered STN: 19 Jul 2002  
TI Pim kinase-related methods for treatment of an allergic response, asthma,  
and transplant rejection  
IN Rothman, Paul; Chen, Peter  
PA The Trustees of Columbia University In the  
City of New York, USA  
SO PCT Int. Appl., 47 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
IC ICM C07D  
CC 1-7 (Pharmacology)

## FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002055489	A2	20020718	WO 2001-US50535	20011227
	WO 2002055489	A3	20021003		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
	US 2004109868	A1	20040610	US 2004-250380	20040120
PRAI	US 2000-258421P	P	20001227		
	WO 2001-US50535	W	20011227		

## CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
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WO 2002055489	ICM	C07D
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AB The invention provides methods for treating an allergic response, asthma, and the onset of transplant rejection in a subject. The methods involve administering an agent which increases the amount and/or the activity of a Pim kinase. The invention also provides a method for determining whether an agent increases the phosphorylation of a Socs-1 protein by a Pim kinase.

ST Pim kinase modulator allergy asthma treatment; transplant rejection treatment Pim kinase modulator; SOCS1 phosphorylation Pim kinase modulator screening

IT Animal cell line  
(293T; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Allergy  
Allergy inhibitors  
Antiasthmatics  
Asthma  
Drug screening  
Inflammation  
Macrophage

Pain

Pruritus

Signal transduction, biological

Swelling, biological

Transplant and Transplantation

Transplant rejection

Urticaria

(Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Interleukin 4

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Nucleic acids

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(SOCS-1 (suppressor of cytokine signaling-1); Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(SOCS-2 (suppressor of cytokine signaling-2); Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transcription factors

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(STAT6 (signal transducer and activator of transcription 6); Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(and small mols.; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(bone marrow; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(bone; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(brain; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Bone

(cell, transplant; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(elongin BC; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(heart; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(intestine; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(kidney; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(liver; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(lung; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(muscle; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(pancreas; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation

(pancreatic islet; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Lymphocyte  
(plasma cell, transplant; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Phosphorylation, biological  
(protein; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Skin  
(redness; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Transplant and Transplantation  
(skin; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Thymus gland  
(thymocyte; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT Blood cell  
Bone  
Bone marrow  
Brain  
Cartilage  
Eye  
Heart  
Intestine  
Kidney  
Liver  
Lung  
Muscle  
Ovary  
Pancreas  
Pancreatic islet of Langerhans  
Skin  
Stem cell  
Stomach  
(transplant; Pim kinase-related methods for treatment of allergic response, asthma, and transplant rejection)

IT 50812-37-8D, Glutathione-S-transferase, Pim-2 kinase fusion products  
144376-45-4, Pim-1 protein kinase 270086-00-5, Pim-3 kinase  
420790-04-1, Pim-2 protein kinase 420790-04-1D, Pim-2 protein kinase,  
GST fusion products  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(Pim kinase-related methods for treatment of allergic response, asthma,  
and transplant rejection)

L9 ANSWER 3 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN  
AN 2002:449525 HCAPLUS  
DN 137:32066  
ED Entered STN: 14 Jun 2002  
TI Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8  
latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter  
and monoclonal antibodies for diagnosis and therapy  
IN Chang, Yuan; Moore, Patrick S.  
PA The Trustees of Columbia University in the  
City of New York, USA  
SO PCT Int. Appl., 111 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
IC ICM A61K039-12  
ICS A61K039-245; C12P021-06; C07H021-04  
CC 15-3 (Immunochemistry)  
Section cross-reference(s): 3, 9, 10, 63

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002045744	A1	20020613	WO 2001-US47217	20011207
W:	AB, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, BG, KZ, MD, RU, TJ, TM, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, CA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
RW:				

US 2002137020	A1	20020926	US 2000-733728	20001208
US 6653465	B2	20031125		
AU 2002026019	A5	20020618	AU 2002-26019	20011207
PRAI US 2000-733728	A	20001208		
WO 2001-US47217	W	20011207		

## CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
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WO 2002045744	ICM	A61K039-12
	ICS	A61K039-245; C12P021-06; C07H021-04
US 2002137020	ECLA	C07K014/03

AB This invention provides an isolated nucleic acid which encodes a Kaposi's sarcoma-associated herpesvirus latency-associated nuclear antigen 2 polypeptide (LANA2) or a fragment thereof and also provides the LANA2 polypeptide. This invention provides an isolated nucleic acid comprising consecutive nucleotides having the sequence of a promoter of Kaposi's sarcoma-associated herpesvirus latency-associated nuclear antigen 2 transcription. This invention also provides a method of inhibiting p53 mediated apoptosis of a cell and a method of producing an antibody which comprises introducing into a cell a replicable vector of the subject invention.

ST Kaposi's sarcoma assoc herpesvirus latency nuclear antigen 2; human herpesvirus 8 LANA2 promoter monoclonal antibody

IT Lymph node, disease  
(Castleman's; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Animal tissue culture  
B cell (lymphocyte)  
Blood analysis  
Blood plasma  
Blood serum  
Canis familiaris  
Cavia porcellus  
Cerebrospinal fluid  
Coliphage .lambda.  
Colorimetric indicators  
Cosmids  
DNA sequences  
Embryo, animal  
Escherichia coli  
Eubacteria  
Eukaryota  
Fluorescent substances  
Gamete and Germ cell

Hematopoietic precursor cell

Human  
Human herpesvirus 8  
Immunoassay  
Labels  
Luminescent substances  
Lymph node  
Lymphocyte  
Mammalia  
Mus  
Mustela putorius furo  
Nucleic acid hybridization  
Oryctolagus cuniculus  
Ovis aries  
Plasmids  
Primates  
Protein sequences  
Rattus  
Reptilia  
Spleen  
Tonsil  
Urine analysis  
YAC (yeast artificial chromosome)

(Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT p53 (protein)  
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)  
(Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)



- IT Antibodies and Immunoglobulins  
 RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)  
 (Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Promoter (genetic element)  
 RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT DNA  
 Gene, microbial  
 Nucleic acids  
 RNA  
 Radionuclides, biological studies  
 Toxins  
 cDNA  
 mRNA  
 RL: BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Antigens  
 Proteins  
 RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)  
 (LANA2; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Drug delivery systems  
 (carriers; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Bone marrow  
 (cells; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Test kits  
 (diagnostic; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Immunoassay  
 (enzyme-linked immunosorbent assay; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Immunoassay  
 (fluorescence; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Cell  
 (host; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Antibodies and Immunoglobulins  
 RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)  
 (humanized; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Immunoassay  
 (immunoblotting; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)
- IT Drug delivery systems  
 (immunconjugates; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide,

polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Diagnosis  
(immunodiagnosis; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Drug delivery systems  
(immunotoxins; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Skin, disease  
(lesion; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Antibodies and Immunoglobulins  
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)  
(monoclonal; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Apoptosis  
(p53-mediated; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Lymphoma  
(primary effusion; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Diagnosis  
(radiodiagnosis; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Genetic vectors  
(replicable; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Diagnosis  
(serodiagnosis; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Animal cell  
(somatic; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Animal  
(transgenic; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT Body fluid  
(transudates and exudates; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT 437131-44-7P  
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)  
(amino acid sequence; Kaposi's sarcoma-associated herpesvirus or human herpesvirus 8 latency-associated nuclear antigen 2 polypeptide, polynucleotide, promoter and monoclonal antibodies for diagnosis and therapy)

IT 437131-45-8P 437131-46-9P  
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)  
(nucleotide sequence; Kaposi's sarcoma-associated herpesvirus or human

herpesvirus 8 latency-associated nuclear antigen 2 polypeptide,  
polynucleotide, promoter and monoclonal antibodies for diagnosis and  
therapy)

IT 437132-47-3 437132-48-4 437132-49-5 437132-50-8 437132-51-9  
437132-52-0

RL: PRP (Properties)

(unclaimed sequence; kaposi's sarcoma-associated herpesvirus or human  
herpesvirus 8 latency-associated nuclear antigen 2 polypeptide,  
polynucleotide, promoter and monoclonal antibodies for diagnosis and  
therapy)

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

- (1) Ballestas; Science 1999, V284, P641 HCAPLUS
- (2) Dittmer; Journal of Virology 1998, V72(10), P8309 HCAPLUS
- (3) Dupin; PNAS 1999, V96, P4546 HCAPLUS
- (4) Friberg; Nature 1999, V402(23), P889
- (5) Katano; Virology 2000, V269, P335 HCAPLUS
- (6) Neipel; Journal of Virology 1997, V71(6), P4187 HCAPLUS
- (7) Russo; PNAS 1996, V93, P14862 HCAPLUS

L9 ANSWER 4 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

AN 1999:614258 HCAPLUS

DN 131:227652

ED Entered STN: 26 Sep 1999

TI Human monoclonal antibodies from tetroma cells

IN Trakht, Ilya

PA The Trustees of Columbia University in the

City of New York, USA

SO PCT Int. Appl., 86 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM G01N033-53

ICS G01N033-567; C07K016-00; A61K039-395; A61K039-42

CC 15-1 (Immunochemistry)

Section cross-reference(s): 1, 8, 14, 63

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9947929	A1	19990923	WO 1999-US5828	19990318
W: AU, CA, JP, MX, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6197582	B1	20010306	US 1998-40833	19980318
CA 2323681	AA	19990923	CA 1999-2323681	19990318
AU 9931889	A1	19991011	AU 1999-31889	19990318
EP 1064551	A1	20010103	EP 1999-913925	19990318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002507398	T2	20020312	JP 2000-537073	19990318
PRAI US 1998-40833	A2	19980318		
WO 1999-US5828	W	19990318		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9947929	ICM	G01N033-53
	ICS	G01N033-567; C07K016-00; A61K039-395; A61K039-42
AB	The author discloses the preparation of antibody-non-producing heteromyeloma and trioma cells from the fusion of human and mouse myeloma and human lymphoid cells, resp. The trioma cell fusion partner, when again fused with a human lymphoid cell, provides a tetroma capable of producing a monoclonal antibody having specific binding affinity for antigen. The invention thus provides a method of producing a monoclonal antibody with specificity for cells, tissue, or disease state. The author also discloses therapeutic and diagnostic application of these tetroma-derived monoclonal antibodies.	
ST	heteromyeloma trioma tetroma monoclonal antibody	
IT	Hybridoma (B-cell, heteromyeloma; for fusion with lymphoid cells in production of monoclonal antibodies)	
IT	Animal cell line (B6B11; for fusion with lymphoid cells in production of monoclonal antibodies)	
IT	Immunoglobulins RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES	

Searched by Noble Jarrell

(Uses)  
 (M, monoclonal; to breast and prostate cancer antigens)

IT Animal cell line  
 (MFP-2; for fusion with lymphoid cells in production of monoclonal antibodies)

IT Transplant rejection  
 (allotransplant; tetroma-derived monoclonal antibodies as therapy for)

IT Toxins  
 RL: ADV (Adverse effect, including toxicity); ANT (Analyte); ANST (Analytical study); BIOL (Biological study)  
 (anthrax; tetroma production of monoclonal antibodies to)

IT Antibodies  
 RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)  
 (autoantibodies; tetroma-derived monoclonal antibodies as therapy against)

IT Thyroid gland, disease  
 (autoimmune thyroiditis; tetroma-derived monoclonal antibodies as therapy for)

IT Magnetic materials  
 (beads, conjugates with tetroma-derived monoclonal antibodies; for disease diagnosis)

IT Diagnosis  
 (cancer; by tetroma-derived monoclonal antibodies to tumor antigens)

IT Chromophores  
 Fluorescent dyes  
 (conjugates with tetroma-derived monoclonal antibodies; for disease diagnosis)

IT Cytotoxic agents  
 (conjugates with tetroma-derived monoclonal antibodies; therapeutic application of)

IT Enzymes, biological studies  
 RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (conjugates, with tetroma-derived monoclonal antibodies; for disease diagnosis)

IT Metabolism, animal  
 (disorder, enzyme dysfunction; tetroma-derived monoclonal antibodies as therapy for)

IT Hormones, animal, biological studies  
 RL: BSU (Biological study, unclassified); BIOL (Biological study)  
 (dysfunction; tetroma-derived monoclonal antibodies as therapy for)

IT Lymphocyte  
 (for fusion with heteromyeloma or trioma in production of monoclonal antibodies)

IT Multiple myeloma  
 (for fusion with lymphoid cells in production of monoclonal antibodies)

IT B cell (lymphocyte)  
 Erythroblast  
 Macrophage  
 Monocyte  
 T cell (lymphocyte)  
 (for fusion with trioma in production of monoclonal antibodies)

IT Gene, animal  
 RL: BSU (Biological study, unclassified); BIOL (Biological study)  
 (for monoclonal antibody heavy and light chains from tetroma)

IT Gel electrophoresis  
 Gel permeation chromatography  
 (for separation of tetroma-derived monoclonal antibody from cognate antigen)

IT Transplant and Transplantation  
 (graft-vs.-host reaction; tetroma-derived monoclonal antibodies as therapy for)

IT Thyroid gland, neoplasm  
 Thyroid gland, neoplasm  
 (inhibitors; tetroma-derived monoclonal antibodies as)

IT Drug delivery systems  
 (liposomes; as carriers of tetroma-derived monoclonal antibodies)

IT Animal cell  
 (mammalian; tetroma production of monoclonal antibodies to antigens specific to)

IT Antitumor agents  
 (mammary gland; tetroma-derived monoclonal antibodies as)

IT Antibodies  
 RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (monoclonal, labeled, tetroma-derived; for diagnostic and therapeutic applications)

IT Antibodies  
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)  
(monoclonal; fusion of human lymphoid cells with heteromyeloma and trioma for production of)

IT Mammary gland  
Mammary gland  
Prostate gland  
Prostate gland  
(neoplasm, inhibitors; tetroma-derived monoclonal antibodies as)

IT Immune complexes  
RL: ANT (Analyte); ANST (Analytical study)  
(of antigens with tetroma-derived monoclonal antibodies)

IT Immunotherapy  
(of autoimmune disease with tetroma-derived monoclonal antibodies)

IT Cell fusion  
(of heteromyeloma and trioma with human lymphoid cells in production of monoclonal antibodies)

IT Cryopreservation  
(of human lymphoid cells for fusion with heteromyeloma and trioma in production of monoclonal antibodies)

IT Antigens  
RL: ANT (Analyte); ANST (Analytical study)  
(preparation of tetroma and production of monoclonal antibodies to)

IT Antitumor agents  
(prostate gland; tetroma-derived monoclonal antibodies as)

IT Shock (circulatory collapse)  
(septic; tetroma-derived monoclonal antibodies as therapy for)

IT Venoms  
(snake; tetroma-derived monoclonal antibodies as therapy against)

IT Venoms  
(spider; tetroma-derived monoclonal antibodies as therapy against)

IT Spleen  
(splenocyte; for fusion with heteromyeloma or trioma in production of monoclonal antibodies)

IT Lupus erythematosus  
(systemic; tetroma-derived monoclonal antibodies as therapy for)

IT Toxins  
RL: ADV (Adverse effect, including toxicity); ANT (Analyte); ANST (Analytical study); BIOL (Biological study)  
(tetanus; tetroma production of monoclonal antibodies to)

IT Animal virus  
Bacteria (Eubacteria)  
Escherichia coli  
Eukaryote (Eukaryotae)  
Fungi  
Insect (Insecta)  
Klebsiella  
(tetroma production of monoclonal antibodies to)

IT Enzymes, analysis  
Immunoglobulins  
Nucleic acids  
RL: ANT (Analyte); ANST (Analytical study)  
(tetroma production of monoclonal antibodies to)

IT Animal cell  
Animal tissue  
Neoplasm  
(tetroma production of monoclonal antibodies to antigens specific to)

IT Anti-AIDS agents  
Antibacterial agents  
Antirheumatic agents  
Antiviral agents  
Fungicides  
(tetroma-derived monoclonal antibodies as)

IT Bacillus anthracis  
Cryptococcus (fungus)  
Ebola virus  
Hantavirus  
Herpesviridae  
Human T-lymphotropic virus 1  
Human T-lymphotropic virus 2  
Human papillomavirus  
Influenza virus  
Staphylococcus  
Streptococcus

(tetroma-derived monoclonal antibodies as therapy against)

IT Autoimmune disease  
Sepsis  
Septicemia  
(tetroma-derived monoclonal antibodies as therapy for)

IT CD3 (antigen)  
CD4 (antigen)  
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)  
(tetroma-derived monoclonal antibodies as therapy for immune  
dysfunction mediated by)

IT Immunoassay  
(tetroma-derived monoclonal antibodies in)

IT Hybridoma  
(tetroma; production of monoclonal antibodies by)

IT Antitumor agents  
Antitumor agents  
(thyroid; tetroma-derived monoclonal antibodies as)

IT Hybridoma  
(trioma; for fusion with lymphoid cells in production of monoclonal  
antibodies)

IT Antigens  
RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL  
(Biological study); USES (Uses)  
(tumor-associated; preparation of tetroma and production of monoclonal antibodies  
to)

IT Spider  
(venom; tetroma-derived monoclonal antibodies as therapy against)

IT Radionuclides, biological studies  
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical  
study); BIOL (Biological study); USES (Uses)  
(with tetroma-derived monoclonal antibodies; for disease diagnosis)

IT 107231-12-9, Botulin  
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)  
(tetroma-derived monoclonal antibodies as therapy against)

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

- (1) Larrick; US 5001065 A 1991 HCAPLUS  
(2) Teng, N; Proc Natl Acad Sci USA December 1983, V80, P7308 MEDLINE

L9 ANSWER 5 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN  
AN 1998:1558 HCAPLUS  
DN 128:58320  
ED Entered STN: 02 Jan 1998  
TI Human radioresistance/cell cycle progression homolog of  
Schizosaccharomyces pombe RAD9  
IN Lieberman, Howard B.; Hopkins, Kevin M.  
PA Trustees of Columbia University, USA; Lieberman, Howard B.;  
Hopkins, Kevin M.  
SO PCT Int. Appl., 56 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
IC ICM C12N  
CC 3-3 (Biochemical Genetics)  
Section cross-reference(s): 1, 6, 13

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9746661	A2	19971211	WO 1997-US8798	19970509
W: AU, CA, JP, MX, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5882862	A	19990316	US 1996-644034	19960509
CA 2261637	AA	19971211	CA 1997-2261637	19970509
AU 9746431	A1	19980105	AU 1997-46431	19970509
PRAI US 1996-644034	A2	19960509		
WO 1997-US8798	W	19970509		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9746661	ICM	C12N
US 5882862	ECLA	C07K014/47A26; C12Q001/68M; C12Q001/68M6B

AB This invention provides isolated nucleic acids which encode a wild-type and a mutant human homolog of RAD9. This invention also provides purified and mutant wild-type human homologs of RAD9. The wild-type human homolog of the fission yeast RAD9 checkpoint control gene was purified from a human placental cDNA library by in situ plaque hybridization. Sequence

anal. showed the full-length clone to have a 1176-bp coding region for a protein of 391 amino acids, which is 26% identical and 52% similar at the amino acid level to S. pombe Rad9. Human RAD9 was localized to chromosomal region 11q13.1-11q13.2. A plasmid vector designated pHRAD9-1 was made by cleaving DNA which encodes a wild-type human RAD9 homolog with HindIII and NotI, creating blunt ends and inserting the DNA into the SmaI site of pART1. This invention also provides methods for determining whether a subject has radiosensitivity; for predicting the effect of radiation therapy or chemotherapy on a subject; for detecting whether a subject has a predisposition to cancer; for treating a subject who is radiosensitive and for preventing or treating cancer in a subject who is radiosensitive. This invention further provides a method of detecting cervical carcinoma in a subject.

- ST human gene RAD9 sequence radiosensitivity chemotherapy; cervical cancer detection human gene RAD9; cell cycle gene RAD9 human
- IT Gene, animal
  - RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
  - (RAD9; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Nervous system
  - (ataxia telangiectasia, treatment of; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Diagnosis
  - (cancer, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Uterus, neoplasm
  - (cervix, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Amniotic fluid
  - Blood analysis
  - Brain, neoplasm
  - Chemotherapy
  - Lung, neoplasm
  - Melanoma
  - Radiotherapy
  - Susceptibility (genetic)
    - (determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Neoplasm
  - (diagnosis, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Embryo, animal
  - (fetus, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Muscle
  - (fiber, gene therapy for cancer radiation therapy or chemotherapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT cDNA sequences
  - (for human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Proteins, specific or class
  - RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
  - (gene RAD9; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Bone marrow
  - Gamete and Germ cell
  - Intestine
  - Liver
    - (gene therapy for cancer radiation therapy or chemotherapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Chromosome
  - (human 13, human RAD9 homolog mapping to human chromosome 11q13; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)
- IT Genetic mapping

(human RAD9 homolog mapping to human chromosome 11q13; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Antitumor agents  
Cell cycle  
Cosmids  
Gene therapy  
Genetic vectors  
Virus vectors  
YAC (yeast artificial chromosome)  
(human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Mammary gland  
(neoplasm, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Protein sequences  
(of human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Plasmid vectors  
(pHRAD9-1; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Diagnosis  
(prenatal, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Kidney, neoplasm  
(renal cell carcinoma, determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Gamma ray  
UV radiation  
(sensitivity; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Cell  
(stem, gene therapy for cancer radiation therapy or chemotherapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Mammal (Mammalia)  
Mouse  
(transgenic, for radiotherapy and chemotherapy sensitivity research; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT Skin, disease  
(xeroderma pigmentosum, treatment of; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT 184594-71-6P, Protein (human brain gene HRAD9)  
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)  
(amino acid sequence; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT 127-07-1, Hydroxyurea  
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(determining radiosensitivity and tumor diagnosis and therapy; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

IT 184595-34-4P, GenBank U53174  
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)  
(nucleotide sequence; human radioresistance/cell cycle progression homolog of Schizosaccharomyces pombe RAD9)

L9 ANSWER 6 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN  
AN 1994:646298 HCAPLUS  
DN 121:246298  
ED Entered STN: 26 Nov 1994  
TI Anhydroretinol and derivatives thereof as antagonists of retinol and 14-hydroxyretroretinol  
IN Hammerling, Ulrich; Buck, Jochen; Derguini, Fadila; Nakanishi, Koji  
PA Sloan-Kettering Institute for Cancer Research, USA; Trustees of Columbia University in the City of New York  
SO PCT Int. Appl., 73 pp.  
CODEN: PIXXD2



DT Patent  
 LA English  
 IC ICM A61K031-015  
 ICS A61K031-045; A61K031-75; A61K031-22; A61K031-23  
 CC 1-6 (Pharmacology)  
 Section cross-reference(s): 15

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9420081	A1	19940915	WO 1994-US2450	19940308
	W: AU, CA, FI, HU, JP, KR, NO, NZ, PL, RO, RU				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	AU 9463614	A1	19940926	AU 1994-63614	19940308
PRAI	US 1993-27880		19930308		
	WO 1994-US2450		19940308		

CLASS

	PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
	WO 9420081	ICM	A61K031-015
		ICS	A61K031-045; A61K031-75; A61K031-22; A61K031-23
AB	This invention provides methods of inhibiting the growth of cells, of treating a subject having a disease characterized by an uncontrolled growth of cells and of blocking an immune response as well as related inflammatory responses in a subject which comprises administering anhydroretinol and derivs. Anhydroretinol antagonizes retinol and 14-hydroxyretroretinol. All-trans anhydroretinol was an antagonist and reversible inhibitor of human B lymphoblastoid tumor cell proliferation. Retro retinyl Me ether reversed the growth-promoting effect of retinol in activated mouse T lymphocytes at 10 <sup>-5</sup> -10 <sup>-8</sup> M.		
ST	anhydroretinol antagonist retinol hydroxyretroretinol; cell proliferation inhibitor anhydroretinol; immune response inhibitor anhydroretinol; antiinflammatory anhydroretinol; neoplasm inhibitor anhydroretinol		
IT	Allergy inhibitors Antidiabetics and Hypoglycemics Cell proliferation Immunosuppressants Inflammation inhibitors Neoplasm inhibitors (anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	<b>Dermatitis</b> (inhibition of chemical; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	Venoms (inhibition of contact allergy to insect; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	Insect (inhibition of contact allergy to venom of; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	Poison ivy Poison oak Poison sumac (inhibition of contact allergy to; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	Monocyte (inhibition of immune response mediated by; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	Autoimmune disease Kidney, disease <b>Psoriasis</b> (treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	Lymphocyte (B-cell, transformed; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		
IT	Neoplasm inhibitors (B-cell lymphoma, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)		

- response and inflammation)
- IT Antigens  
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)  
(CD4, T-cells pos. for; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Antigens  
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)  
(CD8, T-cells pos. for; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Proteins, specific or class  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(CRBP-I (cellular retinol-binding protein I), anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Lymphocyte  
(T-cell, activated; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Inflammation inhibitors  
(antirheumatics, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Immunity  
(cell-mediated, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Allergy inhibitors  
(contact, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Immunity  
(disorder, treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Transplant and Transplantation  
(graft-vs.-host reaction, treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Immunity  
(humoral, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Neoplasm inhibitors  
(leukemia, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Neoplasm inhibitors  
(mammary gland, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Neoplasm inhibitors  
(myelogenous leukemia, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Hematopoietic precursor cell  
(myeloid, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Mammary gland  
(neoplasm, inhibitors, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Proteins, specific or class  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(retinol-binding, anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)
- IT Thyroid gland, disease  
(thyroiditis, treatment; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and

immune response and inflammation)

IT Bone marrow  
Organ  
(transplant, immune response to; anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT 68-26-8, Retinol 153219-51-3  
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT 1224-78-8P, Anhydroretinol  
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)  
(anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

IT 158730-82-6  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(anhydroretinol and derivs. as antagonists of retinol and 14-hydroxyretroretinol for inhibition of cell growth and immune response and inflammation)

=> b wpix

FILE 'WPIX' ENTERED AT 14:22:47 ON 21 OCT 2004  
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FILE LAST UPDATED: 19 OCT 2004 <20041019/UP>  
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HIT STRUCTURES WITHIN THE BIBLIOGRAPHIC DOCUMENT <<<

=> d all l18 tot

L18 ANSWER 1 OF 4 WPIX COPYRIGHT 2004 THE THOMSON CORP on STN

AN 2004-122909 [12] WPIX

DNC C2004-049553

TI Inducing differentiation of embryonic stem cells,  
useful for treating neurodegenerative diseases, comprises contacting the  
cells with rostralizing and/or caudalizing and dorsalizing or ventralizing  
embryonic signaling factors.

DC B04 D16

IN JESSEL, T M; LIEBERAM, I; WICHTERLE, H

PA (UYCO) UNIV COLUMBIA NEW YORK

CYC 101

PI WO 2004007665 A2 20040122 (200412)\* EN 76 C12N000-00

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS  
LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK  
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR  
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT

RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM  
ZW

ADT WO 2004007665 A2 WO 2003-US20399 20030626  
PRAI US 2002-196882 20020716  
IC ICM C12N000-00  
AB WO2004007665 A UPAB: 20040418

NOVELTY - Inducing differentiation of an embryonic stem cell into a differentiated neural cell by contacting the embryonic stem cell with amounts of a rostralizing and/or caudalizing embryonic signaling factor and a dorsalizing or ventralizing embryonic signaling factor effective to produce a differentiated neural cell, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:  
(1) producing differentiated neural cells;  
(2) differentiated neural cells or neural progenitor cells produced by the methods cited above;  
(3) repopulating a spinal cord in a subject;  
(4) treating nervous tissue degeneration in a subject;  
(5) a transgenic non-human animal line containing the embryonic stem cells cited above, where the cells express enhanced green fluorescent protein (eGFP);  
(6) isolating a population of differentiated neural cells; and  
(7) identifying an agent for use in treating a condition associated with motor neuron degeneration.

ACTIVITY - Neuroprotective; Vulnerary; Muscular-Gen.; Nootropic; Anticonvulsant; Antiparkinsonian; Cerebroprotective.  
No biological data given.

MECHANISM OF ACTION - Cell therapy.

USE - The method is useful in inducing differentiation of embryonic stem cells. The differentiated neural cells or the neural progenitor cells are used in analyzing neuron development, function and death, or in monitoring synaptic differentiation (claimed). The cells and the methods may also be used in treating nervous tissue degeneration, such as a peripheral neuropathy or a neurodegenerative disease, or in identifying agents for use in treating a condition associated with neuron degeneration. Diseases that may be treated include amyotrophic lateral sclerosis, neural trauma, paraneoplastic syndrome, polio, postpolio syndrome, progressive bulbar palsy, spinal muscular atrophy, Alzheimer's disease, Binswanger's disease, Huntington's chorea, multiple sclerosis, myasthenia gravis, Parkinson's disease or Pick's disease.

Dwg.0/10

FS CPI  
FA AB; DCN  
MC CPI: B03-A; B04-F0200E; B10-C04A; B11-C08E1; B12-K04E; B14-G02D; B14-J01A3; B14-J01A4; B14-J05; B14-N17B; B14-S01; D05-H08; D05-H09; D05-H14B2

L18 ANSWER 2 OF 4 WPIX COPYRIGHT 2004 THE THOMSON CORP on STN  
AN 2004-098824 [10] WPIX  
DNC C2004-040709  
TI Inducing differentiation of embryonic stem cells into differentiated neural cells, by contacting stem cells with rostralizing and/or caudalizing embryonic signaling factor and dorsalizing or ventralizing signaling factor.

DC B04 D16 P25  
IN JESSEL, T M; LIEBERAM, ; WICHTERLE, H; JESSELL, T M; LIEBERAM, I  
PA (UYCO) UNIV COLUMBIA NEW YORK; (JESS-I) JESSELL T M; (LIEB-I) LIEBERAM I; (WICH-I) WICHTERLE H

CYC 102  
PI US 2004014210 A1 20040122 (200410)\* 34 C12N005-06  
WO 2004007665 A2 20040122 (200412) EN 76 C12N000-00

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS  
LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK  
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR  
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT  
RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM  
ZW

AU 2003247809 A1 20040202 (200453) A47B083-02  
ADT US 2004014210 A1 US 2002-196882 20020716; WO 2004007665 A2 WO 2003-US20399 20030626; AU 2003247809 A1 AU 2003-247809 20030626  
FDT AU 2003247809 A1 Based on WO 2004007665  
PRAI US 2002-196882 20020716  
IC ICM A47B083-02; C12N000-00; C12N005-06  
ICS A47B003-14; A61K031-203; A61K038-17; C12N005-08  
AB US2004014210 A UPAB: 20040901

NOVELTY - Inducing (M1) differentiation of an embryonic stem cell into a differentiated neural cell, by contacting the embryonic stem cell with a rostralizing and/or caudalizing embryonic signaling factor and a dorsalizing or ventralizing embryonic signaling factor, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) a differentiated neural cell (I) produced by (M1);
- (2) producing (M2) differentiated neural cells, by obtaining or generating a culture of embryonic stem cells, contacting the culture embryonic stem cells with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells, contacting the neural progenitor cells with dorsalizing or ventralizing embryonic signaling factor to produce differentiated neural cells, and optionally contacting the differentiated neural cells with at least one neurotrophic factor;
- (3) a population of cells (II) comprising differentiated neural cells produced by M2;
- (4) repopulating (M3) a spinal cord in a subject, by preparing differentiated neural cells using method M2, and transplanting the cells into the spinal chord of the subject;
- (5) treating (M4) nervous tissue degeneration in a subject by transplanting cells prepared according to M2 into the subject;
- (6) neural progenitor cells (NP) produced by obtaining or generating a culture of embryonic stem cells, contacting the culture of embryonic stem cells with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells, and optionally contacting the neural progenitor cells with a dorsalizing or ventralizing embryonic signaling factor;
- (7) differentiated neural cells (III) produced by obtaining or generating a culture of embryonic stem cells, contacting the culture of embryonic stem cells with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells, contacting the neural progenitor cells with dorsalizing or ventralizing embryonic signaling factor to produce differentiated neural cells, and optionally contacting the differentiated neural cells with at least one neurotrophic factor;
- (8) a transgenic non-human animal line containing (III), where the cells express enhanced green fluorescent protein (eGFP);
- (9) isolating a population of differentiated neural cells, involves obtaining or generating a culture of embryonic stem cells that express eGFP, contacting the culture of embryonic stem cells with rostralizing and/or caudalizing embryonic signaling factor to produce neural progenitor cells that express eGFP, contacting neural progenitor cells with dorsalizing or ventralizing embryonic signaling factor to produce differentiated neural cells that express eGFP, optionally contacting the differentiated neural cells with at least one neurotrophic factor, detecting expression of eGFP in the differentiated neural cells, and isolating the differentiated neural cells that express eGFP; and
- (10) identifying an agent for use in treating a condition associated with motor neuron degeneration, by obtaining or generating a culture of embryonic stem cell, contacting the culture of embryonic stem cells with retinoic acid to produce neuronal progenitor cells, activating a Hedgehog signaling pathway in the neuronal progenitor cells to produce motor neurons, where some or all of the motor neurons are degenerated, contacting the degenerated motor neurons with the candidate agent and determining if the agent enhances regeneration of some or all of the degenerated motor neurons.

ACTIVITY - CNS-Gen.; Neuroprotective; Cerebroprotective; Vulnerary; Nootropic; Anticonvulsant; Antiparkinsonian; Muscular-Gen.

No biological data given.

MECHANISM OF ACTION - Cell therapy.

USE - M1 is useful for inducing differentiation of an embryonic stem cell into a differentiated neural cell. The embryonic stem cell is murine embryonic stem cell or human embryonic stem cell. The differentiated neural cell is transplanted into the spinal cord of the subject. The subject is an embryo or human. The subject has nervous tissue degeneration. The nervous tissue degeneration is a peripheral neuropathy or a neurodegenerative disease. The peripheral neuropathy is associated with a condition chosen from amyotrophic lateral sclerosis (ALS), neural trauma, paraneoplastic syndrome, polio, postpolio syndrome, progressive bulbar palsy, and spinal muscular atrophy (SMA), preferably ALS or SMA. The neurodegenerative disease is chosen from Alzheimer's disease, amyotrophic lateral sclerosis (Lou Gehrig's disease), Binswanger's

disease, Huntington's chorea, multiple sclerosis, myasthenia gravis, Parkinson's disease, and Pick's disease. NP and (III) is useful or analyzing neuron development, function and death, or in monitoring synaptic differentiation (all claimed).

Dwg. 0/10

FS CPI GMPI

FA AB; DCN

MC CPI: B03-A; B04-F02; B04-F0200E; B11-C07B3; B11-C08E1; B12-K04A5; B12-K04E; B14-G02D; B14-J01A3; B14-J01A4; B14-J05; B14-N16; B14-N17B; B14-S01; D05-H08; D05-H09; D05-H14B2

L18 ANSWER 3 OF 4 WPIX COPYRIGHT 2004 THE THOMSON CORP on STN

AN 2000-687622 [67] WPIX

DNC C2000-209364

TI Prevention and treatment of contact dermatitis, hyperpigmentation, cutaneous inflammation and other conditions, comprises inhibiting the stem cell factor signaling pathway.

DC B04 D16 D21

IN LONGLEY, B J

PA (UYCO) UNIV COLUMBIA NEW YORK; (LONG-I) LONGLEY B J

CYC 90

PI WO 2000067794 A1 20001116 (200067)\* EN 72 A61K039-395

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL

OA PT SD SE SL SZ TZ UG ZW

W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES

FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS

LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL

TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000048253 A 20001121 (200112) A61K039-395

US 2002123031 A1 20020905 (200260) C12Q001-00

US 6576812 B1 20030610 (200340) G01N033-00

ADT WO 2000067794 A1 WO 2000-US12405 20000505; AU 2000048253 A AU 2000-48253 20000505; US 2002123031 A1 CIP of US 1999-306143 19990506, US 1999-474478 19991229; US 6576812 B1 US 1999-306143 19990506

FDT AU 2000048253 A Based on WO 2000067794

PRAI US 1999-474478 19991229; US 1999-306143 19990506

IC ICM A61K039-395; C12Q001-00; G01N033-00

ICS A01K067-00; A01K067-027; A01K067-033; C07K016-00; C12Q001-70

AB WO 200067794 A UPAB: 20001223

NOVELTY - Preventing or treating diseases comprises administering a compound capable of inhibiting the stem cell factor signaling pathway.

DETAILED DESCRIPTION - Preventing or treating contact dermatitis, hyperpigmentation, asthma, cutaneous inflammation, anaphylaxis or bronchospasm, mastocytosis, urticaria, hypersensitivity, airway inflammation, interstitial cystitis or a tumor which expresses activated kit comprises administering to the subject a compound capable of inhibiting the stem cell factor signaling pathway effective to prevent or treat the conditions.

INDEPENDENT CLAIMS are also included for the following:

(1) providing contraception comprising administering a compound capable of inhibiting the cell factor signaling pathway effective to prevent conception;

(2) desensitizing a subject to an agent comprising administering to the subject, during the afferent phase of an immune response, a compound capable of inhibiting the stem cell factor signaling pathway effective to desensitize the subject;

(3) identifying a composition, compound or procedure which can produce a skin response comprising administering the compound or composition or applying the procedure to transgenic mice which express endogenous epidermal stem cell factor and analyzing the skin of the transgenic mice for a response;

(4) identifying a composition, compound or procedure which can reduce skin response in a subject comprises administering the composition or compound or applying the procedure to the transgenic mice which express endogenous epidermal stem cell factor and which had been induced to produce a skin disease and analyzing the skin to determine the reduction of the skin response;

(5) identifying a compound, composition or procedure which can reduce radiation damage to skin comprises administering the composition or compound or applying the procedure to the skin of the transgenic mice which express endogenous epidermal stem cell factor, subjecting the skin of the transgenic mice and control mice to radiation and analyzing the effects of the composition, compound or procedure on reducing skin radiation damage;

(6) a composition for treating human skin diseases comprising a

compound that can treat skin diseases of the transgenic mice which express endogenous epidermal stem cell factor and a carrier, wherein the compound specifically targets the epidermal stem cell factor or its receptor.

ACTIVITY - Dermatological; antiinflammatory; antiasthmatic; antiinflammatory; antiallergic; immunosuppressive; cytostatic. No biological data is given.

MECHANISM OF ACTION - Stem cell factor signaling pathway inhibitor.

USE - The methods can be used to treat and/or prevent contact dermatitis, hyperpigmentation, asthma, cutaneous inflammation, anaphylaxis or bronchospasm, mastocytosis, urticaria, hypersensitivity, airway inflammation e.g. rhinitis, interstitial cystitis, a tumor which expresses activated kit wherein the tumor is e.g. a gastrointestinal stromal tumor or a germ cell tumor or radiation damage. The method may also be used to provide contraception (claimed).

Dwg.0/13

FS CPI

FA AB; DCN

MC CPI: B04-E01; B04-G01; B04-G21; B04-H16; B04-M01; B04-N04; B11-C08E2; B12-K04E; B14-C03; B14-H01; B14-K01; B14-K01A; B14-N07B; B14-N17; B14-N17C; B14-P01; B14-S06; D05-H09; D05-H11A; D05-H12; D05-H16A

L18 ANSWER 4 OF 4 WPIX COPYRIGHT 2004 THE THOMSON CORP on STN

AN 1999-277439 [23] WPIX

DNC C1999-081536

TI New peptides based on an advanced glycation end product receptor are useful for treating Alzheimer's disease and Down's syndrome.

DC B04 D16

IN LAMSTER, I; SCHMIDT, A M; STERN, D; YAN, S D

PA (UYCO) UNIV COLUMBIA NEW YORK; (LAMS-I) LAMSTER I; (SCHM-I)

SCHMIDT A M; (STER-I) STERN D; (YANS-I) YAN S D

CYC 24

PI WO 9918987 A1 19990422 (199923)\* EN 99 A61K038-00  
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE  
W: AU CA JP MX

AU 9897958 A 19990503 (199937)

EP 1023080 A1 20000802 (200038) EN A61K038-00

R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE

JP 2001519401 W 20011023 (200202) 84 C07K007-04

US 2001053357 A1 20011220 (200206) A61K038-04

US 6555651 B2 20030429 (200331) A61K038-00

ADT WO 9918987 A1 WO 1998-US21346 19981009; AU 9897958 A AU 1998-97958 19981009; EP 1023080 A1 EP 1998-952204 19981009, WO 1998-US21346 19981009; JP 2001519401 W WO 1998-US21346 19981009, JP 2000-515619 19981009; US 2001053357 A1 US 1997-948131 19971009; US 6555651 B2 US 1997-948131 19971009

FDT AU 9897958 A Based on WO 9918987; EP 1023080 A1 Based on WO 9918987; JP 2001519401 W Based on WO 9918987

PRAI US 1997-948131 19971009

IC ICM A61K038-00; A61K038-04; C07K007-04

ICS A61K009-06; A61K009-08; A61K009-12; A61K009-14; A61K039-395;

A61K047-48; A61P001-02; A61P003-10; A61P009-10; A61P015-10;

A61P017-02; A61P021-02; A61P025-28; A61P029-00; A61P035-00;

A61P037-06; A61P043-00; C07K005-00; C07K014-00; C07K014-705;

C07K016-00; C07K017-00; C12P021-08

AB WO 9918987 A UPAB: 19990616

NOVELTY - Novel isolated peptides (I) having an amino acid sequence corresponding to an amino acid sequence of a V-domain of a receptor for an advanced glycation end product (AGEP), are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a composition comprising (I) linked to an antibody or its portion;

(2) a method for inhibiting an amyloid-beta peptide (ABP) interaction with a receptor for AGEP when the receptor is on the surface of a cell, which comprises contacting the cell with an inhibitor of the interaction to inhibit interaction of the ABP with the receptor for AGEP;

(3) a method for inhibiting degeneration of a neuronal cell which comprises contacting the cell with an inhibitor of the interaction of an ABP with a receptor for AGEP so as to inhibit the interaction and thereby inhibit degeneration of the neuronal cell;

(4) a method for inhibiting formation of an ABP fibril on a cell which comprises contacting the cell with an inhibitor of the interaction of an ABP with a receptor for AGEP so as to inhibit the interaction and

thereby inhibit formation of the ABP fibril on a cell;

(5) a method for inhibiting extracellular assembly of an ABP into a fibril which comprises contacting the ABP with an inhibitor of the interaction of an ABP with another ABP so as to inhibit the interaction and thereby inhibit extracellular assembly of an ABP into a fibril;

(6) a method for inhibiting aggregation of ABP on the surface of a cell which comprises contacting the ABP with an inhibitor of the interaction of the ABP with a receptor for AGEF so as to inhibit the interaction and thereby inhibit aggregation of ABP on the surface of a cell;

(7) a method for inhibiting infiltration of a microglial cell into senile plaques which comprises contacting the microglial cell with an inhibitor of the interaction of an ABP with a receptor for AGEF on the surface of the microglial cell, so as to inhibit the interaction and thereby inhibit infiltration of a microglial cell into senile plaques;

(8) a method for inhibiting activation of a microglial cell by an ABP which comprises contacting the microglial cell with an inhibitor of the interaction of the ABP with a receptor for AGEF on the surface of the microglial cell so as to inhibit the interaction and thereby inhibit activation of a microglial cell;

(9) a method for treating a subject with a condition associated with an interaction of an ABP with a receptor for AGEF on a cell, which comprises administering to the subject an inhibitor capable of inhibiting the interaction of the ABP with the receptor for AGEF;

(10) a method for evaluating the ability of an agent to inhibit binding of an ABP with a V-domain of a receptor for AGEF on the surface of a cell which comprises:

(a) contacting the cell with the agent and ABP;

(b) determining the amount of ABP bound to the cell; and

(c) comparing the amount of bound ABP determined in (b) with the amount determined in the absence of the agent, thus evaluating the ability of the agent to inhibit the binding of ABP to the V-domain of the receptor for AGEF on the surface of the cell;

(11) a method for inhibiting activation of a NF-kappaB gene in a cell which comprises contacting the cell with an inhibitor of the interaction of ABP with a receptor for AGEF on the cell so as to inhibit the interaction and thus inhibit activation of NF-kappaB in the cell;

(12) a method for inhibiting periodontal disease in a subject which comprises administering topically to the subject a pharmaceutical composition which comprises soluble receptor for an AGEF (sRAGE) to accelerate wound healing and thereby inhibit periodontal disease;

(13) a method for inhibiting an AGEF's interaction with a receptor for AGEF when the receptor is on the surface of a cell, which comprises contacting the cell with an inhibitor of the interaction to inhibit interaction of the AGEF with the receptor for AGEF;

(14) a method for treating a subject with a condition associated with an interaction of an AGEF with a receptor for AGEF on a cell, which comprises administering to the subject an inhibitor capable of inhibiting the interaction of the AGEF with the receptor for AGEF.

USE - The methods can be used for treating conditions associated with an interaction of an ABP or an AGEF with a receptor for AGEF, e.g. diabetes, Alzheimer's disease, senility, renal failure, hyperlipidemic atherosclerosis, neuronal cytotoxicity, Down's syndrome, dementia associated with head trauma, amyotrophic lateral sclerosis, multiple sclerosis, amyloidosis, an autoimmune disease, inflammation, a tumor, cancer, male impotence, wound healing, periodontal disease, neuropathy, retinopathy, nephropathy or neuronal degeneration (claimed).

Dwg. 0/1

FS CPI

FA AB; DCN

MC CPI: B04-G01; B14-C03; B14-F06; B14-G02D; B14-H01; B14-J01A4; B14-N03;  
B14-N06B; B14-N10; B14-N17B; B14-S01; B14-S04; D05-H10;  
D05-H11

=> b home

FILE 'HOME' ENTERED AT 14:22:57 ON 21 OCT 2004

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(FILE 'HOME' ENTERED AT 14:01:47 ON 21 OCT 2004)

FILE 'HCAPLUS' ENTERED AT 14:01:53 ON 21 OCT 2004

L1           33 E4-6  
               E LONGLEY J/AU  
 L2           12 E3,E9  
 L3           418 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA  
               E SKIN DISEASE/CT  
               E E4+ALL  
               E E2  
               E E3+ALL  
 L4           70942 "SKIN, DISEASE"+OLD,NT/CT  
 L5           11 L4 AND L1-2  
 L6           24 L4 AND L3  
               E STEM CELL/CT  
               E E3+ALL  
 L7           30857 STEM CELL+NT/CT  
               E CELL/CT  
               E E3+ALL  
 L8           11056 CELL+OLD,NT/CT (L) STEM  
 L9           6 L5-6 AND L7-8

FILE 'WPIX' ENTERED AT 14:16:23 ON 21 OCT 2004

L10          48555 (B12-A07 OR C12-A07 OR B14-N17? OR C14-N17?)/MC  
               E STEM CELL#/BI  
               E STEM CELL/BI  
               E STEM CELL/ABEX  
 L11          4846 (STEM (1A) CELL?)/BIX  
 L12          710 L10 AND L11  
               E LONGLEY B/AU  
 L13          3 E4  
 L14          1 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA  
 L15          1 L12 AND L13  
 L16          810 UYCO/PACO  
 L17          4 L12 AND (L14 OR L16)  
 L18          4 L15 OR L17

FILE 'MEDLINE' ENTERED AT 14:28:32 ON 21 OCT 2004

L19          165235 STEM CELLS+NT/CT  
 L20          416171 SKIN DISEASES+NT/CT  
 L21          53892 L19/MAJ  
 L22          333514 L20/MAJ  
 L23          117556 L22 (L) (TH. OR PC)/CT  
 L24          113 L21 AND L23  
               E LONGLEY B/AU  
 L25          47 E4-6  
 L26          49494 (COLUMBIA AND UNIV?)/CS  
 L27          0 L24 AND L25  
 L28          0 L24 AND L27  
 L29          70 L24 AND PY<=1999  
               SEL AN 1-2 9-10 12-13 15-16 19 22 35-37  
 L30          13 E1-13 AND L29  
 L31          0 L24 AND L26

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(FILE 'HOME' ENTERED AT 14:01:47 ON 21 OCT 2004)

(FILE 'HCAPLUS' ENTERED AT 14:01:53 ON 21 OCT 2004)

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E LONGLEY B/AU
L1      33 E4-6
        E LONGLEY J/AU
L2      12 E3,E9
L3      418 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
        E SKIN DISEASE/CT
        E E4+ALL
        E E2
        E E3+ALL
L4      70942 "SKIN, DISEASE"+OLD,NT/CT
L5      11 L4 AND L1-2
L6      24 L4 AND L3
        E STEM CELL/CT
        E E3+ALL
L7      30857 STEM CELL+NT/CT
        E CELL/CT
        E E3+ALL
L8      11056 CELL+OLD,NT/CT (L) STEM
L9      6 L5-6 AND L7-8

FILE 'WPIX' ENTERED AT 14:16:23 ON 21 OCT 2004
L10     48555 (B12-A07 OR C12-A07 OR B14-N17? OR C14-N17?)/MC
        E STEM CELL#/BI
        E STEM CELL/BI
        E STEM CELL/ABEX
L11     4846 (STEM (1A) CELL?)/BIX
L12     710 L10 AND L11
        E LONGLEY B/AU
L13     3 E4
L14     1 (TRUST? AND COLUMBIA AND UNIV?)/CS,PA
L15     1 L12 AND L13
L16     810 UYCO/PACO
L17     4 L12 AND (L14 OR L16)
L18     4 L15 OR L17

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(FILE 'MEDLINE' ENTERED AT 14:28:32 ON 21 OCT 2004)

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L19     165235-STEM CELLS+NT/CT
L20     416171 SKIN DISEASES+NT/CT
L21     53892 L19/MAJ
L22     333514 L20/MAJ
L23     117556 L22 (L) (TH. OR PC)/CT
L24     113 L21 AND L23
        E LONGLEY B/AU
L25     47 E4-6
L26     49494 (COLUMBIA AND UNIV?)/CS
L27     0 L24 AND L25
L28     0 L24 AND L27
L29     70 L24 AND PY<=1999
        SEL AN 1-2 9-10 12-13 15-16 19 22 35-37
L30     13 E1-13 AND L29

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=&gt; b medl

(FILE 'MEDLINE' ENTERED AT 14:51:31 ON 21 OCT 2004)

FILE LAST UPDATED: 20 OCT 2004 (20041020/UP). FILE COVERS 1951 TO DATE.

On February 29, 2004, the 2004 MeSH terms were loaded. See HELP RLOAD for details. OLDMEDLINE now back to 1951.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2004 vocabulary. See <http://www.nlm.nih.gov/mesh/> and [http://www.nlm.nih.gov/pubs/techbull/nd03/nd03\\_mesh.html](http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html) for a description of changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=&gt; d all 130 tot

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L30 ANSWER 1 OF 13 MEDLINE on STN
AN 2001150874 MEDLINE

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Searched by Noble Jarrell

DN PubMed ID: 11225638  
 TI Growth factors secreted by fibroblasts: role in healing diabetic foot ulcers.  
 AU Mansbridge J N; Liu K; Pinney R E; Patch R; Ratcliffe A; Naughton G K  
 CS Advanced Tissue Sciences, Inc., La Jolla, CA 92037, USA..  
 jonathan.mansbridge@advancedtissue.com  
 SO Diabetes, obesity & metabolism, (1999 Sep) 1 (5) 265-79. Ref: 109  
 Journal code: 100883645. ISSN: 1462-8902.  
 CY England: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Priority Journals  
 EM 200103  
 ED Entered STN: 20010404  
 Last Updated on STN: 20010404  
 Entered Medline: 20010315  
 CT Check Tags: Human  
 Animals  
 Cells, Cultured  
 Cytokines: SE, secretion  
 Diabetic Foot: PP, physiopathology  
 \*Diabetic Foot: TH, therapy  
 \*Fibroblasts: PH, physiology  
 Gene Expression  
 Growth Substances: GE, genetics  
 \*Growth Substances: PH, physiology  
 Keratinocytes: PH, physiology  
 Macrophages: PH, physiology  
 Neovascularization, Physiologic  
 Skin: BS, blood supply  
 Skin Transplantation  
 Wound Healing  
 CN 0 (Cytokines); 0 (Growth Substances)  
 L30 ANSWER 2 OF 13 MEDLINE on STN  
 AN 2000427040 MEDLINE  
 DN PubMed ID: 10937098  
 TI Autologous fibroblasts for treatment of facial rhytids and dermal depressions. A pilot study.  
 AU Watson D; Keller G S; Lacombe V; Fodor P B; Rawnsley J; Lask G P  
 CS Division of Head and Neck Surgery, University of California, Los Angeles, USA.  
 SO Archives of facial plastic surgery : official publication for the American Academy of Facial Plastic and Reconstructive Surgery, Inc. and the International Federation of Facial Plastic Surgery Societies, (1999 Jul-Sep) 1 (3) 165-70.  
 Journal code: 100883500. ISSN: 1521-2491.  
 CY United States  
 DT (CLINICAL TRIAL)  
 Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 200009  
 ED Entered STN: 20000922  
 Last Updated on STN: 20000922  
 Entered Medline: 20000908  
 AB OBJECTIVE: To assess effectiveness of intradermal injections of autologous fibroblasts for the treatment of facial rhytids and dermal depressions. DESIGN: Six-month prospective pilot study. Photographs and silicone molds were taken of a prominent rhytid or dermal depression from each patient prior to treatment and at 6 months after treatment. SETTING: Specialty clinic in academic medical center. PATIENTS: Ten adults (age range, 24-69 years) who each exhibited a prominent rhytid or depressed facial scar. INTERVENTION: A 3-mm postauricular skin biopsy specimen from each participant was sent to Isolagen Technologies, Inc, laboratories, where a fibroblast cell line was developed. Three injection sessions were performed at 2-week intervals; target areas were the study site as well as behind the ear. MAIN OUTCOME MEASURES: Subjective improvement scores were obtained by each patient and 2 clinicians at every follow-up visit. Skin surface topographical features were evaluated with optical profilometry by comparing silicone molds before and after injection. Histological analysis was performed on a biopsy specimen of the postauricular injection site. RESULTS: Nine of 10 patients noted a 60% to 100% improvement with

the treatment; clinicians made similar observations. Size reduction of 10% up to 85% of the study site was demonstrated by optical profilometry for every patient. Microscopically, there was evidence of increased thickness and density of dermal-layer collagen. CONCLUSIONS: Intradermal injection of autologous fibroblasts may be an effective treatment option for facial rhytids and depressed scars.

CT Check Tags: Female; Human; Male

Acne Vulgaris: TH, therapy

Adult

Aged

\*Cicatrix: TH, therapy

Esthetics

\*Facial Dermatoses: TH, therapy

\*Fibroblasts

\*Fibroblasts: TR, transplantation

Follow-Up Studies

Injections, Intradermal

Middle Aged

Patient Satisfaction

Pilot Projects

Prospective Studies

Reconstructive Surgical Procedures

Transplantation, Autologous

Treatment Outcome

L30 ANSWER 3 OF 13 MEDLINE on STN

AN 1999116001 MEDLINE

DN PubMed ID: 9916172

TI Three-dimensional fibroblast culture implant for the treatment of diabetic foot ulcers: metabolic activity and therapeutic range.

AU Mansbridge J; Liu K; Patch R; Symons K; Pinney E

CS Advanced Tissue Sciences, Inc., La Jolla, California 92037, USA.

SO Tissue engineering, (1998 Winter) 4 (4) 403-14.

Journal code: 9505538. ISSN: 1076-3279.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199903

ED Entered STN: 19990316

Last Updated on STN: 19990316

Entered Medline: 19990301

AB Dermagraft is three-dimensional, allogeneic, human neonatal dermal fibroblast culture grown on a degradable scaffold and cryopreserved. Clinical trials for treatment of diabetic foot ulcers showed optimal healing within a therapeutic range of metabolic activity, determined by 3[4,5-dimethylthiazol-2-yl]2,5-diphenyltetrazolium bromide (MTT) reduction. Actions of Dermagraft in wound repair include colonization by cells and provision of growth factors and cytokines, both activities dependent on living cells. Cells in the cryopreserved culture showed 60% viability by dye exclusion and, when isolated, were able to proliferate in monolayer culture. Protein synthesis by Dermagraft was inhibited 70-98% by cryopreservation, but, if within the therapeutic range, recovered to 45-85% of the prefreeze value over 48 h. Subtherapeutic Dermagraft showed variable, low recovery. Expression of mRNA for vascular endothelial growth factor (VEGF), platelet-derived growth factor A chain, and insulin-like growth factor-1 was reduced >83% in subtherapeutic compared with therapeutic Dermagraft. Granulocyte colony-stimulating factor and VEGF protein secretion, determined by enzyme-linked immunosorbent assay (ELISA), and angiogenic activity also depended on therapeutic range. VEGF secretion dropped sharply with MTT reductase in subtherapeutic tissue. The data demonstrate the critical dependence of the therapeutic properties of this living dermal implant on recovery of protein synthesis, growth factor expression, and angiogenesis, determined by metabolic activity.

CT Check Tags: Human; Male

Animals

Cell Survival

Cells, Cultured: TR, transplantation

Chick Embryo

Cryopreservation

Culture Media, Conditioned: CH, chemistry

\*Diabetic Foot: SU, surgery

Endothelial Growth Factors: BI, biosynthesis

Enzyme-Linked Immunosorbent Assay

Fibroblasts: ME, metabolism

\*Fibroblasts: TR, transplantation

Granulocyte Colony-Stimulating Factor: BI, biosynthesis  
 Infant, Newborn  
 Lymphokines: BI, biosynthesis  
 Neovascularization, Physiologic  
 RNA, Messenger: BI, biosynthesis  
 Skin, Artificial  
 Transplantation, Homologous  
 Vascular Endothelial Growth Factor A  
 Vascular Endothelial Growth Factors  
 Wound Healing

RN 143011-72-7 (Granulocyte Colony-Stimulating Factor)  
 CN 0 (Culture Media, Conditioned); 0 (Endothelial Growth Factors); 0  
 (Lymphokines); 0 (RNA, Messenger); 0 (Vascular Endothelial Growth Factor  
 A); 0 (Vascular Endothelial Growth Factors)

L30 ANSWER 4 OF 13 MEDLINE on STN  
 AN 1999113022 MEDLINE  
 DN PubMed ID: 9893171  
 TI Studies on the biological activity of the dermal regeneration template.  
 AU Yannas I V  
 CS Departments of Mechanical Engineering and Materials Science and  
 Engineering, Massachusetts Institute of Technology, Cambridge, Mass  
 02139-4307, USA.  
 SO Wound repair and regeneration : official publication of the Wound Healing  
 Society [and] European Tissue Repair Society, (1998 Nov-Dec) 6  
 (6) 518-23. Ref: 42  
 Journal code: 9310939. ISSN: 1067-1927.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Priority Journals  
 EM 199902  
 ED Entered STN: 19990311  
 Last Updated on STN: 19990311  
 Entered Medline: 19990222

AB In mammals, the skin normally responds to injury by the processes of wound  
 contraction and scar formation. However, if a unique acellular dermal  
 regeneration template is placed into the wound, wound contraction is  
 strongly inhibited, and the skin undergoes a regenerative-type healing.  
 It is proposed that these phenomena result from the disruption of a  
 "mechanically coherent" organization of myofibroblasts within the wound.  
 Evidence suggests that the dermal regeneration template prevents the  
 formation of cell-extracellular matrix interactions (fibronexi), which  
 foster the assemblage of a mechanically coherent wound bed. Additional  
 studies support a similar mechanism for the enhanced regenerative-like  
 response to peripheral nerves when extracellular matrix tubes are used to  
 facilitate healing.

CT Check Tags: Human  
 Adult  
 Animals  
 Cicatrix: PP, physiopathology  
 \*Cicatrix: PC, prevention & control  
 Contracture: PP, physiopathology  
 \*Contracture: PC, prevention & control  
 Extracellular Matrix: PH, physiology  
 \*Fibroblasts: PH, physiology  
 \*Nerve Regeneration: PH, physiology  
 \*Skin Physiology  
 Templates, Genetic  
 \*Wound Healing: PH, physiology

L30 ANSWER 5 OF 13 MEDLINE on STN  
 AN 1998312640 MEDLINE  
 DN PubMed ID: 9650620  
 TI Corrective transduction of human epidermal stem cells in  
 laminin-5-dependent junctional epidermolysis bullosa.  
 AU Dellambra E; Vailly J; Pellegrini G; Bondanza S; Golisano O; Macchia C;  
 Zambruno G; Meneguzzi G; De Luca M  
 CS Laboratory of Tissue Engineering, I.D.I.-IRCCS, Istituto Dermopatico  
 dell'Immacolata, Rome, Italy.  
 SO Human gene therapy, (1998 Jun 10) 9 (9) 1359-70.  
 Journal code: 9008950. ISSN: 1043-0342.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)

LA English  
 FS Priority Journals  
 EM 199809  
 ED Entered STN: 19980925  
 Last Updated on STN: 19980925  
 Entered Medline: 19980916

AB Laminin-5 is composed of three distinct polypeptides, alpha3, beta3, and gamma2, which are encoded by three different genes, LAMA3, LAMB3, and LAMC2, respectively. We have isolated epidermal keratinocytes from a patient presenting with a lethal form of junctional epidermolysis bullosa characterized by a homozygous mutation of the LAMB3 gene, which led to complete absence of the beta3 polypeptide. In vitro, beta3-null keratinocytes were unable to synthesize laminin-5 and to assemble hemidesmosomes, maintained the impairment of their adhesive properties, and displayed a decrease of their colony-forming ability. A retroviral construct expressing a human beta3 cDNA was used to transduce primary beta3-null keratinocytes. Clonogenic beta3-null keratinocytes were transduced with an efficiency of 100%. Beta3-transduced keratinocytes were able to synthesize and secrete mature heterotrimeric laminin-5. Gene correction fully restored the keratinocyte adhesion machinery, including the capacity of proper hemidesmosomal assembly, and prevented the loss of the colony-forming ability, suggesting a direct link between adhesion to laminin-5 and keratinocyte proliferative capacity. Clonal analysis demonstrated that holoclones expressed the transgene permanently, suggesting stable correction of epidermal stem cells. Because cultured keratinocytes are used routinely to make autologous grafts for patients suffering from large skin or mucosal defects, the full phenotypic reversion of primary human epidermal stem cells defective for a structural protein opens new perspectives in the long-term treatment of genodermatoses.

CT Check Tags: Human; Support, Non-U.S. Gov't  
 Animals  
 Cells, Cultured  
 DNA: AN, analysis  
 Desmosomes: ME, metabolism  
 \*Epidermis: CY, cytology  
 Epidermolysis Bullosa, Junctional: GE, genetics  
 Epidermolysis Bullosa, Junctional: PA, pathology  
 \*Epidermolysis Bullosa, Junctional: TH, therapy  
 Fluorescent Antibody Technique  
 \*Gene Therapy  
 Genetic Vectors  
 Infant, Newborn  
 Keratinocytes: CY, cytology  
 Keratinocytes: UL, ultrastructure  
 Laminin: BI, biosynthesis  
 \*Laminin: GE, genetics  
 Mice  
 Precipitin Tests  
 RNA: AN, analysis  
 Retroviridae: GE, genetics  
 \*Stem Cells: CY, cytology  
 \*Transduction, Genetic

RN 63231-63-0 (RNA); 9007-49-2 (DNA)  
 CN 0 (Genetic Vectors); 0 (Laminin)

L30 ANSWER 6 OF 13 MEDLINE on STN  
 AN 1998271201 MEDLINE  
 DN PubMed ID: 9608274  
 TI [Prospects of use human fetal fibroblasts in the treatment of various etiology wounds].  
 Perspektivy ispol'zovaniia fetal'nykh fibroblastov cheloveka pri lechenii ran razlichnoi etiologii.  
 AU Kolokol'tseva T D; Iurchenko N D; Kolosov N G; Nechaeva E A; Shumakova O V; Khristo S A  
 SO Vestnik Rossiiskoi akademii meditsinskikh nauk / Rossiiskaia akademiia meditsinskikh nauk, (1998) (3) 32-5.  
 Journal code: 9215641. ISSN: 0869-6047.  
 CY RUSSIA: Russian Federation  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA Russian  
 FS Priority Journals  
 EM 199806  
 ED Entered STN: 19980708  
 Last Updated on STN: 19980708  
 Entered Medline: 19980622

AB The investigations have demonstrated a high efficiency of the certified human fetal lung fibroblasts in the treatment of skin wounds of various etiology. For treatment, a fibroblast monolayer grown on a backing was applied to various types of wounds (burns frostbites, donor site suppurations) and in shin phlegmon. There was wound debrediment and rapid skin recovery. Employing the cells from the certified cell culture banks provides not only a biologically and genetic uniform material, but also makes it possible to standardize treatments which are useful for wide medical application, by delivering the standard cells to medical institutions which have no experience in culturing. To set up banks of cell cultures and to store them for a long time at a temperature of liquid nitrogen in adequate quantities of ampoules allow the cultured fibroblasts to be provided with if a large quantity of cells is required, which may be useful in emergencies.

CT Check Tags: Human  
 Cell Division  
 Cell Transplantation  
 Cells, Cultured  
 English Abstract  
 \*Fetal Tissue Transplantation  
 Fibroblasts: CY, cytology  
 \*Fibroblasts: TR, transplantation  
 Lung: CY, cytology  
 Lung: EM, embryology  
 \*Skin Diseases: TH, therapy  
 Treatment Outcome  
 Wound Healing  
 \*Wounds and Injuries: TH, therapy

L30 ANSWER 7 OF 13 MEDLINE on STN  
 AN 1998180327 MEDLINE  
 DN PubMed ID: 9521032  
 TI New skin for old: developments in biological skin substitutes.  
 CM Comment on: Arch Dermatol. 1998 Mar;134(3):293-300. PubMed ID: 9521027  
 AU Phillips T J  
 SO Archives of dermatology, (1998 Mar) 134 (3) 344-9.  
 Journal code: 0372433. ISSN: 0003-987X.  
 CY United States  
 DT Commentary  
 Editorial  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199804  
 ED Entered STN: 19980416  
 Last Updated on STN: 19980416  
 Entered Medline: 19980409

CT Check Tags: Human  
 Cells, Cultured  
 \*Fibroblasts: TR, transplantation  
 Graft Rejection  
 \*Keratinocytes: TR, transplantation  
 Risk Factors  
 Skin Transplantation  
 \*Skin, Artificial  
 Skin, Artificial: AE, adverse effects  
 \*Varicose Ulcer: TH, therapy

L30 ANSWER 8 OF 13 MEDLINE on STN  
 AN 1998180321 MEDLINE  
 DN PubMed ID: 9521027  
 TI Rapid healing of venous ulcers and lack of clinical rejection with an allogeneic cultured human skin equivalent. Human Skin Equivalent Investigators Group.  
 CM Comment in: Arch Dermatol. 1998 Mar;134(3):344-9. PubMed ID: 9521032  
 Comment in: Arch Dermatol. 1998 Nov;134(11):1483-4. PubMed ID: 9828891  
 AU Falanga V; Margolis D; Alvarez O; Auletta M; Maggiasimo F; Altman M; Jensen J; Sabolinski M; Hardin-Young J  
 CS Department of Dermatology and Cutaneous Surgery, University of Miami School of Medicine, Fla 33136, USA.  
 SO Archives of dermatology, (1998 Mar) 134 (3) 293-300.  
 Journal code: 0372433. ISSN: 0003-987X.  
 CY United States  
 DT (CLINICAL TRIAL)  
 Journal; Article; (JOURNAL ARTICLE)  
 (MULTICENTER STUDY)  
 (RANDOMIZED CONTROLLED TRIAL)

LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199804  
 ED Entered STN: 19980416  
 Last Updated on STN: 20000303  
 Entered Medline: 19980409

AB OBJECTIVE: To test the safety, efficacy, and immunological impact of a cultured allogeneic human skin equivalent (HSE) in the treatment of venous ulcers. DESIGN: Prospective, randomized study. SETTING: Multicenter study in the outpatient setting. INTERVENTION: Each patient with a venous ulcer received either compression therapy alone or compression therapy and treatment with HSE. The patients were evaluated for HSE safety, complete (100%) ulcer healing, time to wound closure, wound recurrence, and immune response to the HSE. OUTCOME: The study was completed as planned in 293 randomized patients. RESULTS: Treatment with HSE was more effective than compression therapy in the percentage of patients healed by 6 months (63% vs 49%;  $P=.02$ , Fisher exact test, 2-tailed) and the median time to complete wound closure (61 days vs 181 days;  $P=.003$ , log-rank test). Treatment with HSE was superior to compression therapy in healing larger ( $> 1000 \text{ mm}^2$ ;  $P=.02$ ) and deeper ulcers ( $P=.003$ ) and ulcers of more than 6 months' duration ( $P=.001$ ). Occurrence of adverse events was similar in both groups. No symptoms or signs of rejection occurred in response to treatment with HSE, and no HSE-specific immune responses were detected in vitro to bovine collagen or to alloantigens expressed on keratinocytes or fibroblasts. CONCLUSIONS: Treatment with HSE healed venous ulcers more rapidly and in more patients than compression therapy alone. There was no clinical or laboratory evidence of rejection or sensitization in response to HSE application. These data suggest that HSE represents a significant advance in the treatment of venous ulcers, particularly those that are difficult to heal.

CT Check Tags: Female; Human; Male; Support, Non-U.S. Gov't  
 Aged  
 Animals  
 Antibodies: AN, analysis  
 Bandages  
 Cattle  
 Collagen: IM, immunology  
 Cytotoxicity, Immunologic  
 \*Fibroblasts: TR, transplantation  
 Graft Rejection  
 HLA Antigens: IM, immunology  
 \*Keratinocytes: TR, transplantation  
 Prospective Studies  
 Recurrence  
 \*Skin, Artificial  
 \*Varicose Ulcer: TH, therapy  
 Wound Healing

RN 9007-34-5 (Collagen)  
 CN 0 (Antibodies); 0 (HLA Antigens)

L30 ANSWER 9 OF 13 MEDLINE on STN  
 AN 97420209 MEDLINE  
 DN PubMed ID: 9274713  
 TI The importance of epidermal stem cells in keratinocyte-mediated gene therapy.  
 AU De Luca M; Pellegrini G  
 SO Gene therapy, (1997 May) 4 (5) 381-3.  
 Journal code: 9421525. ISSN: 0969-7128.  
 CY ENGLAND: United Kingdom  
 DT Editorial  
 LA English  
 FS Priority Journals  
 EM 199709  
 ED Entered STN: 19971008  
 Last Updated on STN: 19971008  
 Entered Medline: 19970919

CT Check Tags: Human  
 \*Burns: TH, therapy  
 Clone Cells  
 \*Epidermis: CY, cytology  
 \*Epidermolysis Bullosa: TH, therapy  
 \*Gene Therapy: MT, methods  
 Interleukin-6: GE, genetics  
 \*Keratinocytes  
 Skin Transplantation  
 \*Stem Cells



Transduction, Genetic  
 CN 0 (Interleukin-6)

L30 ANSWER 10 OF 13 MEDLINE on STN  
 AN 97182109 MEDLINE  
 DN PubMed ID: 9030156  
 TI Single exposures to 5-fluorouracil: a possible mode of targeted therapy to reduce contractile scarring in the injured tendon.  
 AU Khan U; Occleston N L; Khaw P T; McGrouther D A  
 CS Department of Plastic and Reconstructive Surgery, University College, London, England.  
 SO Plastic and reconstructive surgery, (1997 Feb) 99 (2) 465-71.  
 Journal code: 1306050. ISSN: 0032-1052.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199703  
 ED Entered STN: 19970407  
 Last Updated on STN: 19970407  
 Entered Medline: 19970324

AB After injury, adhesions may develop between the digital flexor tendons and their sheaths. Fibroblasts are key cells in this fibrotic adhesive process, and two possible sources for these cells are the synovial sheath and the endotenon tissue (tendon core). Fibroblasts seeded into a collagen lattice will contract the collagen. This fibroblast-populated collagen lattice contraction was used to investigate the ability of the fibroblasts from the synovial sheath and endotenon to reorganize collagen (an important function in the formation of adhesions). Endotenon and synovial fibroblasts isolated from 30 animals were used in the study. Synovial fibroblasts produced significantly greater collagen lattice contraction compared with endotenon fibroblasts ( $p < 0.05$ ). The possibility of preventing collagen lattice contraction with a single, nontoxic 5-minute treatment of the fibroblast-populated collagen lattices with the antimetabolite 5-fluorouracil was investigated. Compared with controls the degree of fibroblast-populated collagen lattice contraction was significantly inhibited ( $p < 0.05$ ) with the use of 5-fluorouracil for endotenon and synovial cells. These results demonstrate the potential for locally targeted therapy in tendon healing. Because of the different contractile properties of the two cell lines, a change in the balance between intrinsic and extrinsic healing might be achieved with this method of therapy; in turn, this might lead to better functional results following surgery.

CT Check Tags: Support, Non-U.S. Gov't  
 Animals  
 \*Antimetabolites: AD, administration & dosage  
 Cells, Cultured  
 Cicatrix: ET, etiology  
 \*Cicatrix: PC, prevention & control  
 \*Collagen: DE, drug effects  
 \*Collagen: PH, physiology  
 Cytoskeleton: DE, drug effects  
 \*Fibroblasts: DE, drug effects  
 Fibroblasts: PH, physiology  
 \*Fluorouracil: AD, administration & dosage  
 Rabbits  
 \*Tendon Injuries: CO, complications  
 Tendon Injuries: PA, pathology

RN 51-21-8 (Fluorouracil); 9007-34-5 (Collagen)  
 CN 0 (Antimetabolites)

L30 ANSWER 11 OF 13 MEDLINE on STN  
 AN 94171837 MEDLINE  
 DN PubMed ID: 8126023  
 TI Effects of hyaluronan on collagen fibrillar matrix contraction by fibroblasts.  
 AU Huang-Lee L L; Wu J H; Nimni M E  
 CS Department of Biochemistry, School of Medicine, University of Southern California, Los Angeles.  
 SO Journal of biomedical materials research, (1994 Jan) 28 (1) 123-32.  
 Journal code: 0112726. ISSN: 0021-9304.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals

EM 199404  
ED Entered STN: 19940420  
Last Updated on STN: 19940420  
Entered Medline: 19940412

AB Hyaluronan, found in high concentrations in fetal tissues, appears to have a major role in preventing scar formation in fetal wounds. Nevertheless, its role in inhibiting wound contractures associated with scar formation has not been clearly demonstrated. Our current study evaluated the effects of hyaluronan using an in vitro floating collagen fibrillar matrix (CFM) contraction model. The results demonstrated that the contraction of CFM by fibroblasts was significantly reduced when high concentrations (> 1 mg/mL) of hyaluronan were present in the media. This phenomenon is unique to hyaluronan, because chondroitin sulfate was ineffective in this connection. Fibroblast migration and proliferation studies indicated that high concentrations of hyaluronan stimulated cell migration and had no cytotoxic effects. Some possible mechanisms by which high concentrations of hyaluronan reduced CFM contraction by fibroblasts were proposed. Because the viscosity of a hyaluronan solution is much greater than that of chondroitin sulfate, and this increases with concentration, we investigated whether this property in itself was an important factor in inhibiting CFM contraction. No direct correlation was found between the viscosity of glycosaminoglycans and their ability to reduce CFM contraction.

CT Check Tags: Human  
Animals  
Cattle  
Cell Division: DE, drug effects  
Cell Movement: PH, physiology  
Cells, Cultured  
Chondroitin Sulfates: PD, pharmacology  
\*Cicatrix: PC, prevention & control  
\*Collagen: DE, drug effects  
DNA: ME, metabolism  
\*Fibroblasts: DE, drug effects  
Fibroblasts: UL, ultrastructure  
Glycosaminoglycans: CH, chemistry  
Glycosaminoglycans: IP, isolation & purification  
\*Hyaluronic Acid: PD, pharmacology  
Viscosity

RN 9004-61-9 (Hyaluronic Acid); 9007-28-7 (Chondroitin Sulfates); 9007-34-5 (Collagen); 9007-49-2 (DNA)

CN 0 (Glycosaminoglycans)

L30 ANSWER 12 OF 13 MEDLINE on STN  
AN 94096931 MEDLINE  
DN PubMed ID: 8271751  
TI [Treatment of chronic non-healing donor sites: transplantation of cultured human fibroblasts].  
Lechenie dlitelno ne zazhivaiushchikh donorskikh uchastkov: transplan tatsiia kultivirovannykh allofibroblastov cheloveka.

AU Moroz V Iu; Grishkevich V M; Alekseev A A; Glushchenko E V; Sarkisov D S; Gurukov Sh R; Tumanov V P; Morozov S S

SO Khirurgiia, (1993 Jul) (7) 71-5.  
Journal code: 0412765. ISSN: 0023-1207.

CY RUSSIA: Russian Federation  
DT Journal; Article; (JOURNAL ARTICLE)  
LA Russian  
FS Priority Journals  
EM 199402  
ED Entered STN: 19940215  
Last Updated on STN: 19940215  
Entered Medline: 19940203

AB The authors discuss the first results of the use of cultivated human allofibroblasts in the treatment of persisting wounds of the donor areas in 13 patients with deep burns and cicatricial deformities caused by the burns. Transplantation of cell cultures led to complete epithelialization of the wounds in 6-10 days. Complication, suppuration of the grafted material, was recorded in one case. Reaction of graft rejection was not encountered. The authors believe that the effect of the transplantation is due to stimulation of the epithelialization processes by the cultivated fibroblasts.

CT Check Tags: Human  
Burns: CO, complications  
\*Burns: SU, surgery  
Cells, Cultured  
Chronic Disease

Cicatrix: ET, etiology  
 Cicatrix: PP, physiopathology  
 \*Cicatrix: SU, surgery  
 English Abstract  
 \*Fibroblasts: TR, transplantation  
 Reoperation  
 \*Skin Transplantation  
 Treatment Outcome  
 Wound Healing

L30 ANSWER 13 OF 13 MEDLINE on STN  
 AN 94058308 MEDLINE  
 DN PubMed ID: 8239704  
 TI Keratinocyte gene therapy.  
 AU Vogel J C  
 CS Dermatology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Md.  
 SO Archives of dermatology, (1993 Nov) 129 (11) 1478-83. Ref: 52  
 Journal code: 0372433. ISSN: 0003-987X.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199312  
 ED Entered STN: 19940117  
 Last Updated on STN: 19940117  
 Entered Medline: 19931214  
 AB BACKGROUND: Gene therapy is currently being used in clinical trials to treat a variety of diseases. In keratinocyte gene therapy, the gene that will correct the disease by expressing the normal protein or enzyme is inserted and expressed in keratinocytes. Keratinocytes have significant potential, as a target cell of gene therapy, in the treatment of both systemic diseases as well as skin diseases caused by a genetic defect in keratinocytes. OBSERVATIONS: Although keratinocyte gene therapy is not yet being tested in clinical trials, animal models do exist where keratinocytes are being used to secrete factors such as human growth hormone and factor IX (for hemophilia) into the systemic circulation. Genetic diseases of the skin such as recessive epidermolysis bullosa dystrophica or xeroderma pigmentosum have not yet been treated with keratinocyte gene therapy in animal models. CONCLUSIONS: Keratinocytes have many advantages as a target cell in gene therapy, and progress has been made using animal models. However, the sustained and efficient delivery of factors to the bloodstream by keratinocytes expressing a transgene has not yet been accomplished. Future goals are to obtain adequate levels of the desired factors, hormones, or enzymes for sustained periods of time, either in keratinocytes or in the vascular system.  
 CT Check Tags: Human  
 Animals  
 Basement Membrane: ME, metabolism  
 Factor IX: ME, metabolism  
 \*Fibroblasts  
 Forecasting  
 Gene Expression  
 \*Gene Therapy: MT, methods  
 Genetic Vectors  
 Growth Hormone: BL, blood  
 \*Keratinocytes  
 Models, Biological  
 Mutation  
 Skin Diseases: BL, blood  
 Skin Diseases: GE, genetics  
 \*Skin Diseases: TH, therapy  
 Tissue Culture  
 RN 9001-28-9 (Factor IX); 9002-72-6 (Growth Hormone)  
 CN 0 (Genetic Vectors)

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